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IBM Technical Disclosure Bulletins

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Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB; PLUR=YES; OP=AND			
L4	l1 with l2	3	L4
L3	l1 and L2	59	L3
L2	(treat$ or diminish$ or decreas$ or inhibit$) near6 (tumor or cancer or neoplasia or hyperplasia)	46886	L2
L1	(nucleotide or nucleic adj acid or polynucleotide or dna) near6 (p27 or kip or kip1)	82	L1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 3 of 3 returned.

1. 6589505 . 14 Jan 00; 08 Jul 03. Cells that lack p19ink4d and p27kip1 activity and methods of use thereof. Roussel; Martine F., et al. 424/9.2; 424/9.1 435/320.1 435/325 435/375 800/14 800/3 800/8 800/9. A61K049/00 C12N005/00 C12N015/00 C12P021/00 A01K067/00 A01K067/027.

2. 6409664 . 24 Nov 99; 25 Jun 02. Nomograms to aid in the treatment of prostatic cancer. Kattan; Michael W., et al. 600/300; 128/898. A61B005/00 A61B019/00.

3. 6245965 . 29 Jan 99; 12 Jun 01. Knockout mice and cells that lack p19INK4d and p27KIP1 activity and methods of use thereof. Roussel; Martine F., et al. 800/18; 435/325 800/13 800/14 800/3 800/8. G01N033/00 A01K067/00 C12N015/85 C12N015/86.

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Terms	Documents
l1 with l2	3

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     (FILE 'HOME' ENTERED AT 16:19:49 ON 03 SEP 2003)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 16:20:00 ON 03 SEP
     2003
L1           194 S (NUCLEOTIDE OR NUCLEIC(W)ACID OR POLYNUCLEOTIDE OR DNA)(5A)(
L2        451569 S (TREAT? OR INHIBIT? OR DIMINISH? OR DECREAS?)(6A)(TUMOR OR CA
L3            11 S L1 AND L2
L4             9 DUP REM L3 (2 DUPLICATES REMOVED)

=> d bib ab 1-9 l4

L4   ANSWER 1 OF 9  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
     DUPLICATE 1
AN   2003:267573  BIOSIS
DN   PREV200300267573
TI   p27Kip1 inhibition of GRB2-SOS formation can regulate Ras activation.
AU   Moeller, Stephanie J.; Head, Elizabeth D.; Sheaff, Robert J. (1)
CS   (1) University of Minnesota Cancer Center, 420 Delaware St. SE, MMC 806,
     Minneapolis, MN, 55455, USA: sheaf004@tc.umn.edu USA
SO   Molecular and Cellular Biology, (June 2003, 2003) Vol. 23, No. 11, pp.
     3735-3752. print.
     ISSN: 0270-7306.
DT   Article
LA   English
AB   p27Kip1 (p27) is often inappropriately downregulated in aggressive human
     cancers. Although p27 can inhibit cyclin-dependent
     kinases (CDKs), low p27 does not always correlate with increased CDK
     activity. Furthermore, cells derived from p27-/- mice respond to
     antimitogens, maintain restriction point control, and do not deregulate
     CDKs. Thus, disruption of a p27 function other than CDK inhibition may
     contribute to the disease state. A yeast two-hybrid screen identified
     growth factor receptor-bound protein 2 (GRB2) as a p27 binding partner. We
     now demonstrate that p27 can inhibit GRB2 function by blocking its
     association with the guanine nucleotide exchange factor SOS.
     Endogenous p27 is rapidly exported from the nucleus to the
     cytoplasm in response to mitogen stimulation, where it binds GRB2
     concomitant with a decrease in GRB2-associated SOS. As predicted,
     mitogen-stimulated p27-/- cells maintained their GRB2-SOS complexes for
     significantly longer. The Ras/mitogen-activated protein kinase pathway
     does not appear to be deregulated in cells lacking p27 despite excess
     GRB2-SOS, suggesting that additional control mechanisms are present. A
     transient-transfection approach was employed to show that p27 can inhibit
     Ras activation by targeting GRB2 and further revealed that the CDK and
     GRB2 inhibitory functions of p27 are separable and distinct. Thus, p27
     downregulation may compromise control of Ras, one of the most common
     oncogenic events in human cancer.

L4   ANSWER 2 OF 9  CAPLUS  COPYRIGHT 2003 ACS on STN
AN   2002:90060  CAPLUS
DN   136:146233
TI   Nucleic acids comprising regions of the rat PEG-3 (progression elevated
     gene-3) promoter and uses thereof
IN   Fisher, Paul B.; Su, Zao-Zhong
PA   The Trustees of Columbia University in the City of New York, USA
SO   PCT Int. Appl., 90 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.         KIND  DATE          APPLICATION NO.  DATE
     ---------------    ----  --------      ---------------  --------
PI   WO 2002008242      A1    20020131      WO 2001-US23099  20010720
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W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1309603 A1 20030514 EP 2001-954874 20010720
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRAI US 2000-621781 A 20000721
WO 2001-US23099 W 20010720

AB This invention provides for an isolated nucleic acid comprising a PEG-3 (progression elevated gene-3) promoter comprising the nucleotide sequence beginning with the guanosine (G) at position -270 and ending with the cytosine (C) at position +194 of SEQ ID NO: 1. The invention also provides for a method for identifying an agent which modulates PEG-3 promoter activity in a cell. The invention also provides for a method for **treating cancer** in a subject by specific expression of the promoter linked to a gene of interest in cancerous cells such that regulated expression from the promoter results in growth suppression or cell death. Subtractive hybridization expts. identified rat gene PEG-3 as a gene which is expressed in transformed rat embryo cells, a model for tumor progression. Addnl. mRNA and protein expression data for gene PEG-3 from several rat cell lines with different phenotypes for tumor progression showed a correlation between gene PEG-3 expression level and transformation progression. The 5'-flanking region of rat gene PEG-3 has been characterized, including identification of the transcription start site, involvement of transcription factor AP1 and PEA3 binding sites in promoter activity, correlation between transcription factors cJun and PEA3 binding and promoter activity, and ability of co-transfected transcription factors cJun and PEA3 to cooperatively activate a promoter-reporter gene construct.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
AN 2002:244412 CAPLUS
DN 136:261315
TI Genetic diagnosis of malignant tumor predisposition with **p27** gene single **nucleotide** polymorphism
IN Takahashi, Hiroyuki; Okano, Hironao; Darnell, Robert B.
PA Japan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2002095484	A2	20020402	JP 2000-291869	20000926
PRAI JP 2000-291869		20000926		

AB A genetic diagnosis or screening method for malignant tumor risk factors by anal. of p27kip1 gene single nucleotide polymorphism (SNP), located on human chromosome 12, is disclosed. C/C polymorphism at the -79 position was found to assocd. with higher incidence of breast cancer, non-small cell lung cancer, prostate cancer, and large intestine cancer. T/T allele, on the other hand was assocd. with higher incidence of ovarian cancer and lung small-cell carcinoma.

L4 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
AN 2003:247624 CAPLUS

DN 138:249812
TI Human 9.9-kDa **DNA**-PK interacting protein **KIP** like protein and its cDNA and therapeutic use
IN Mao, Yumin; Xie, Yi
PA Shanghai Biowindow Gene Development Inc., Peop. Rep. China
SO Faming Zhuanli Shenqing Gongkai Shuomingshu, 31 pp.
CODEN: CNXXEV
DT Patent
LA Chinese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	CN 1351066	A	20020529	CN 2000-125835	20001026
PRAI	CN 2000-125835		20001026		

AB The invention provides protein and cDNA sequences for a novel human 9.9-kDa protein cloned from human fetal brain, which shares a similar mRNA expression pattern to DNA-PK (**DNA**-dependent protein kinase) interacting protein **KIP** (kinase interacting protein). Methods of expressing and prepg. the above recombinant protein and its antibody are described. The invention further relates to applications of related gene or protein products for the **treatment** of related diseases, such as **cancer**, blood diseases, HIV infection, immune diseases and inflammation. Methods of screening for related analogs, agonists, inhibitors, and antagonists and using them as therapeutic drugs are also described.

L4 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
AN 2000:900853 CAPLUS
DN 134:39172
TI Markers for prostate cancer
IN Cordon-Cardo, Carlos; Scher, Howard I.; Koff, Andrew
PA Sloan-Kettering Institute for Cancer Research, USA
SO PCT Int. Appl., 128 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000077258	A1	20001221	WO 2000-US16007	20000609
	W: CA, JP, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	EP 1208232	A1	20020529	EP 2000-938256	20000609
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
PRAI	US 1999-329917	A2	19990610		
	WO 2000-US16007	W	20000609		

AB This invention provides a method for detg. the aggressiveness of a prostate carcinoma comprising: (a) obtaining a sample of the prostate carcinoma; and (b) detecting the presence of p27 protein in the prostate carcinoma, the absence of p27 indicating that the prostate carcinoma is aggressive. This invention also provides a method for diagnosing a benign prostate hyperplasia comprising: (a) obtaining an appropriate sample of the hyperplasia; and (b) detecting the presence of the p27 RNA, a decrease of the p27 RNA indicating that the hyperplasia is benign. This invention provides various uses of p27 in prostate cancer. Finally, this invention also provides different marker for prostate cancer. To det. whether loss of p27 expression was a common feature in prostate cancer, 74 prostate carcinomas from primary and metastatic sites, representing different hormone sensitivities were studied by immunohistochem. staining and in situ hybridization. Other markers such as cyclin D1, cyclin-dependent kinase inhibitor p16, and Her-2/neu were also studied.
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 6 OF 9 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AN 2000:222345 SCISEARCH
GA The Genuine Article (R) Number: 293RA
TI The **tumor** growth-**inhibiting** cell adhesion molecule CEACAM1 (C-CAM) is differently expressed in proliferating and quiescent epithelial cells and regulates cell proliferation
AU Singer B B; Scheffrahn I; Obrink B (Reprint)
CS KAROLINSKA INST, MED NOBEL INST, DEPT CELL & MOL BIOL, BOX 285, SE-17177 STOCKHOLM, SWEDEN (Reprint); KAROLINSKA INST, MED NOBEL INST, DEPT CELL & MOL BIOL, SE-17177 STOCKHOLM, SWEDEN
CYA SWEDEN
SO CANCER RESEARCH, (1 MAR 2000) Vol. 60, No. 5, pp. 1236-1244.
Publisher: AMER ASSOC CANCER RESEARCH, PO BOX 11806, BIRMINGHAM, AL 35202.
ISSN: 0008-5472.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 49
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The hemophilic cell adhesion molecule CEACAM1 (C-CAM, BGP, CD66a) occurs as two coexpressed isoforms, CEACAM1-L and CEACAM1-S, in epithelia, endothelia, and leukocytes, CEACAM1-L can **inhibit tumor** growth; this effect is influenced by CEACAM1-S. To characterize the growth regulatory properties of CEACAM1, we analyzed the expression patterns of the isoforms, and here we demonstrate that both the expression levels and the S:L isoform ratios differ in proliferating and quiescent rat epithelial cells. Quiescent prostate NbE cells expressed more CEACAM1 than quiescent bladder NET-II cells, a pattern that correlated with the expression levels in the parental tissues, In contrast, both the expression levels and the isoform ratios were strikingly similar in proliferating NbE and NET-II cells, showing that a particular CEACAM1 expression pattern is compatible with cell proliferation. However, in confluent cells, CEACAM1 seemed to exert inhibitory effects on cell proliferation. Addition of anti-CEACAM1 antibodies to quiescent, confluent cells caused decreased expression of the cyclin-dependent kinase inhibitor, **p27**(Kip1), stimulated growth factor-dependent **DNA** synthesis, and altered the S:L isoform ratio toward the ratio characteristic of proliferating cells. Taken together, our data suggest that CEACAM1 contributes to contact inhibition of cell proliferation in confluent cells but allows proliferation when expressed at different isoform ratios.

L4 ANSWER 7 OF 9 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AN 1999:28637 SCISEARCH
GA The Genuine Article (R) Number: 150LG
TI Analysis of the cyclin-dependent kinase **inhibitor** p27(Kip1) in muscle invasive bladder **cancer**
AU Serth J; Kuczyk M (Reprint); Machtens S; Bokemeyer C; Herrmann R; Hartmann J; Knuchel R; Jonas U
CS UNIV HANNOVER, DEPT UROL, SCH MED, CARL NEUBERG STR 1, D-30625 HANNOVER, GERMANY (Reprint); UNIV HANNOVER, DEPT UROL, SCH MED, D-30625 HANNOVER, GERMANY; UNIV TUBINGEN, DEPT HEMATOL ONCOL, TUBINGEN, GERMANY; UNIV REGENSBURG, DEPT PATHOL, D-8400 REGENSBURG, GERMANY
CYA GERMANY
SO ONCOLOGY REPORTS, (JAN-FEB 1999) Vol. 6, No. 1, pp. 229-233.
Publisher: INT JOURNAL ONCOLOGY, C/O PROFESSOR D A SPANDIDOS, EDITORIAL OFFICE, 1, S MERKOURI ST, ATHENS 116 35, GREECE.
ISSN: 1021-335X.
DT Article; Journal
FS CLIN
LA English
REC Reference Count: 25

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB It has been suggested that a deregulated cell cycle control contributes to the development of human malignancies due to the lass of critical antiproliferative mechanisms. The cell cycle is controlled at two checkpoints, one at the G1-S and another at the G2-M transition. Several genes including the structurally related p21(WAF/CIP1) gene, the downstream mediator of the p53 tumor suppressor gene, and the p27(Kip1) gene have been identified as inducers of cell cycle arrest at the G1 checkpoint when substantial DNA damage has occurred to avoid further replication of the altered genome. Recently, a heat stable 27 kDa protein, the transcript of the p27(Kip1) gene, has been identified and was suggested to substantially participate in cell cycle control at the GI checkpoint. Previous investigations have correlated decreased expression of the p27(Kip1) protein with an increased biological aggressiveness of breast and small cell lung cancer. However, the moleculargenetic analysis of a variety of human malignancies including prostate cancer failed to identify any alteration at the p27(Kip1) gene locus, therefore suggesting a loss of p27(Kip1) protein expression to result from post-transcriptional/posttranslational events or from so far unknown regulatory mechanisms. So far, bladder cancer specimens have neither been investigated for **p27**(Kip1) alterations on the **DNA** level, nor has the result of moleculargenetic analysis been correlated with an immunohistochemically detected expression of the gene product, the p27(Kip1) protein. The present study is the first to describe **p27** (Kip1) gene alterations on the **DNA** level in 3 of 42 muscle invasive bladder cancer specimens. In contrast, loss of p27(Kip1) protein expression was observed in 14 of 42 (33%) tumors. According to the previously reported observation in a variety of human malignancies, in bladder cancer loss of p27(Kip1) protein expression seems to result from post-transcriptional or posttranslational events.

L4 ANSWER 8 OF 9 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
AN 2000:111903 BIOSIS
DN PREV200000111903
TI Inhibition of CDK2, CDK4 and cyclin E and increased expression of p27Kip1 during **treatment** with interferon-alpha in carcinoid **tumor** cells.
AU Zhou, Y.; Wang, S.; Gobl, A.; Oberg, K. (1)
CS (1) Endocrine Oncology Unit of Internal Medicine, Uppsala University Hospital, S-751 85, Uppsala Sweden
SO Journal of Biological Regulators and Homeostatic Agents, (Oct. Dec., 1999) Vol. 13, No. 4, pp. 207-215.
ISSN: 0393-974X.
DT Article
LA English
SL English
AB IFN-alpha has presented antitumor effect in about 50% of carcinoid tumor patients, though the antitumor mechanism of IFN-alpha is still to be elucidated. In this study we demonstrated that IFN-alpha could result in accumulation of S-phase population and upregulation of cyclin-dependent kinase inhibitor (CKI), **p27**. Moreover, IFN-alpha inhibits **DNA** synthesis assessed by (3H) thymidine incorporation and colony formation on soft agar. Immunodepletion of p27 increased CDK2- and cyclin E-associated kinase activities. These data suggest that IFN-alpha exerts its antiproliferative effects in the neuroendocrine differentiated cell lines by: 1) inhibition of DNA synthesis and colony formation, 2) upregulation on the mRNA and protein expressions of the CKI, p27.

L4 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
AN 1995:801649 CAPLUS
DN 123:192373
TI Isolated **p27** protein and its encoding **nucleic acid** molecules

IN Massague, Joah; Roberts, James M.; Koff, Andrew; Polyak, Kornelia
PA Sloan-Kettering Institute for Cancer Research, USA; Fred Hutchinson Cancer Research Center
SO PCT Int. Appl., 129 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9518824	A1	19950713	WO 1995-US247	19950109
	W: AU, CA, FI, HU, JP, KR, NO, NZ, RU				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 5688665	A	19971118	US 1994-275983	19940715
	AU 9515251	A1	19950801	AU 1995-15251	19950109
	AU 699969	B2	19981217		
	EP 749442	A1	19961227	EP 1995-906793	19950109
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 09509311	T2	19970922	JP 1995-518645	19950109
PRAI	US 1994-179045	A	19940107		
	US 1994-275983	A	19940715		
	WO 1995-US247	W	19950109		

AB An isolated protein is provided having an apparent mol. wt. of .apprx.27 kDa and capable of binding to and inhibiting the activation of a cyclin E-Cdk2 complex. The **nucleic acid** encoding the **p27** protein, vectors, and host cells for producing the recombinant form of the p27 protein are also provided. Methods are described for identifying agents capable of modulating the ability of p27 to inhibit the activation of the cyclin E-Cdk2 complex. Finally, subjects diagnosed with hyperproliferative (e.g., cancer or neoplasia) or hypoproliferative (e.g., ulcer) disorders can be treated with these materials. Thus, protein p27Kip1 was purified from arrested Mv1Lu mink epithelial cells and shown to bind to the cyclin E-Cdk2 complex and to inhibit the activation of Cdk2 kinase by cyclin E. Kip1 tryptic peptide sequences were used to design degenerate oligonucleotide primers for cDNA amplification and cloning. The mink clone was incomplete, whereas full-length reading frames were isolated from human (198 amino acids, 22,257 Da) and mouse (197 amino acids, 22,208 Da).

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